

Louisiana State University LSU Digital Commons

LSU Agricultural Experiment Station Reports

LSU AgCenter

1908

A preliminary report on the so-called cerebro-spinal meningitis of horses

Howard Jay Milks

Follow this and additional works at: <http://digitalcommons.lsu.edu/agexp>

Recommended Citation

Milks, Howard Jay, "A preliminary report on the so-called cerebro-spinal meningitis of horses" (1908). *LSU Agricultural Experiment Station Reports*. 613.
<http://digitalcommons.lsu.edu/agexp/613>

This Article is brought to you for free and open access by the LSU AgCenter at LSU Digital Commons. It has been accepted for inclusion in LSU Agricultural Experiment Station Reports by an authorized administrator of LSU Digital Commons. For more information, please contact gcostel@lsu.edu.

AGRICULTURAL EXPERIMENT STATION

OF THE

Louisiana State University
and A. and M. College,

BATON ROUGE.

A Preliminary Report

ON THE SO-CALLED

Cerebro-Spinal Meningitis of Horses.

BY

H. J. MILKS, D. V. M.

THE DAILY STATE, OFFICIAL JOURNAL OF LOUISIANA.
BATON ROUGE:

1908.

A PRELIMINARY REPORT

ON THE SO-CALLED

Cerebro-Spinal Meningitis of Horses.

BY H. J. MILKS.

During the fall of 1906 and the summer of 1907 Cerebro-Spinal Meningitis was very prevalent in certain parts of this State. It is known as: Staggers, Blind Staggers, Sleepy Staggers, Bottom Sickness, etc.

Occurrence—The disease was not confined to any particular locality. It occurred in lowlands, and in the hill and bluff districts. It attacked animals of all ages and, we believe, horses more than mules. The outbreak extended over a large area, but did not usually attack many animals at the same place, although one owner lost all his horses (three) in the course of three or four days. Animals were attacked at rather widely separated places. We have seen it on both sides of a bayou, extending several miles. It was not confined to animals fed upon any one feed, though the majority of those attacked were allowed to graze.

Cause—Numerous theories have been advanced as to the cause of this disease. It has been attributed to grazing upon low, marshy places, hence the name Bottom Sickness. The cause also has been laid to mouldy corn or fodder, poisonous plants, exposure to sun, impure water, etc. Mayo, reporting a very similar disease, concludes it to be due to a fungus, *Aspérgillus glaucus*. The spores enter the circulation, find lodgment in the organs and set up inflammatory conditions. The cerebral symptoms were due to an abscess of the brain. He has also recovered the above fungus from the different organs.

Chester, of the Delaware Station, has carried on a series of feeding experiments with negative results. Some even point out the infectious nature of the disease.

In Louisiana the disease attacked animals upon such a varied diet that it is hard to connect it with any particular food. As

will be seen later from the case reports, the disease attacked animals fed upon hay and oats, hay and corn, oats and grass, hay and grass, and grass alone. To be sure, many of the animals alleged to have had no grass have still had it in certain amounts. One case, that of a foal four or five days old, reported by Dr. Joseph L. Drexler, of Thibadoux, seemed to contract the disease through the milk of its mother. The mother died of the disease a day or so before the foal. The foal showed all symptoms of the disease.

It can hardly be attributed to toxic plants, or a greater number of animals, kept under exactly similar conditions, would have taken the disease instead of such widely separated cases. This also applies to the theory of infection, because it would seem that many more animals would have been affected. However, if due to a micro-organism, that organism must have great difficulty in entering the body, but when once it gains entrance it must be especially active. Furthermore, if due to a micro-organism our methods are at fault or the organism is too small to be observed.

Symptoms—In the mild cases we got dullness, stupor, weakness, hanging of the head, paresis or slight loss of control over one or more limbs, a slight rise in temperature, 102° to 103° F., often difficulty in swallowing. The visible mucosæ were congested and brownish yellow. In these mild cases the weakness never became so great that the animal could not stand and usually it was able to take some nourishment and water.

The more severe cases were manifested by the same general symptoms, often, however, the respirations were much increased and labored. The temperature ran to 104°-106° F. Pulse might be practically normal or accelerated and hard. Opisthotonus was not marked nor often present. They became rapidly weaker, often partially blind, stood with all legs in a bracing attitude and sometimes found support against the side of the stall or fence. Sometimes they were delirious and would stand with the head pressed against the wall, or turn in a circle either to left or right. There was sometimes heat in the region of the poll and coldness of the extremities. Usually the severe cases were unable to stand after 12 to 36 hours, and fell unable to rise again. They then died in a delirium within a few hours or

lay in a comatose condition from 12 to 36 hours. In the delirious cases the temperature usually kept up pretty well, respirations and pulse were also much accelerated, while in the comatose condition the pulse returned to about normal, temperature to nearly normal, and respirations as though the animal was in a deep sleep.

As the disease progressed we occasionally got a fetid discharge from the nose and mouth, due no doubt to decomposition of the secretions retained in those parts.

In the severe cases the animals usually refused food, but often showed a desire for water, although unable to drink. The digestive tract was almost completely paralyzed. Purgatives seemed to do little good, no matter what the dose. The hypodermic use of eserine or arecolene did not produce purgation, but did exhibit other physiological phenomena.

Course and Mortality—The disease generally runs a rapidly fatal course, lasting from a few hours to four or five days—usually not more than three or four days. The time given by some authorities, 8 to 12 days, is entirely too long, except in cases that survive.

In those cases that survived, the disease attacked slowly, the animal usually *taking* some nourishment and showed all the symptoms of a mild attack.

The mortality was 90 per cent or more. Treatment availed little, unless started in the first few hours of the disease, and even then prognosis was unfavorable.

POST-MORTEM APPEARANCES.

Buckley (Maryland Bulletin 53, 1898) noted slight congestion of the stomach in some cases, intestines unchanged, except for dry fecal matter in the posterior part. Spleen and liver unchanged. Kidneys natural in size and color. Heart and lungs usually unchanged, except in case of the latter, when the animal had been down a long time, there might be a slight congestion on the under side. The meninges of the spinal cord and brain showed greater or less degree of congestion. The *septum nasi* was covered with a fetid discharge; its blood vessels were injected.

MacCullum (in Journal Exp. Med., Vol VI, No. 1, November, 1902) made examinations of several brains from the out-

break referred to above and found as follows: Four brains examined; none showed any signs of the presence of an inflammation of the meninges. In the frontal region on each side, anterior to the motor region of the cortex, there was a slightly depressed area, softly fluctuant, but not marked by any superficial hyperæmia or discoloration. On sectioning this brain a glairy fluid with small granular pulpy masses of whitish tissue flowed out from the softened area. The lesion seemed almost entirely limited to the underlying white matter.

Microscopically there was complete destruction of the brain substance, which was partially replaced by a colloid-like material. The blood vessels in an infected area showed a marked inflammatory condition, with exudation of red and white cells into the perilymph space. Hyalin-like material was also found in the blood vessels. These formations were mainly found in the small vessels. In a later outbreak of the disease he was unable to find the lesion of the brain, but did find that of the blood vessels.

Moore, V. A., has been unable to find any changes in the nervous system and other organs that could be detected by gross examination. In one case the above organs were examined microscopically with similar results.

McCarthy and Ravenel found lesions in the upper gastrointestinal tract and in the nervous system. The latter were divided thus:

1. Small round celled peri-capsular infiltration of the inter-vertebral and gasserian ganglia.
2. Congestion of the cerebral and cerebellar cortex and capillary hemorrhage.
3. The choroid plexus was changed into a triangular tumor-like mass, due to a proliferation of the elastic tissue surrounding the vessels.
4. Distinct degenerative changes in the nerves supplying the larynx and neck.

Mayo (Kansas Bulletin 24, September, 1891) says that the circulatory and respiratory systems were normal, except a slight greenish tinge to the mucosæ in some cases. There was more or less irritation to the stomach and small intestines, but not enough to cause serious symptoms. The large intestine usually

contained a large amount of feces. In all cases the liver was congested and sometimes the kidneys also.

In our investigations the post-mortem lesions were based upon five cases. In all cases the blood vessels of the meninges and the brain showed congestion. (Fig. 1.) Otherwise the brain substance and cord appeared normal. Cross sections of the brain and medulla showed much ecchymosis and extravasation of blood. In some cases there was much fluid beneath the dura; in others only a slight amount. No softening or other microscopic lesion could be detected.

The blood was usually dark and did not clot readily. Heart was usually normal, but frequently contained anti-mortem clots. The lungs were normal, except for a slight hypostatic congestion on the under side. The digestive tract was apparently normal, except that the posterior end usually contained much dry fecal matter. In one case there was marked congestion of the last fourteen or sixteen feet of the small intestine. In another case there was slight congestion. However, this condition did not show from the exterior and may have been overlooked in other cases.

The liver usually contained much blood, spleen normal in size and appearance and might contain much blood or be very dry. The kidneys showed slight congestion, but were otherwise normal in appearance. The bladder usually contained apparently normal urine.

Microscopic examinations were made of the brains and kidneys of two horses and the liver, spleen and intestine of one. Briefly stated, there were shrinkage of certain of the cells of the nervous tissue of the brain and inflammatory conditions, especially around the blood vessels. The blood vessels also contained much blood. The kidneys and liver showed some granular degeneration and congestion. In one case the blood vessels of the kidneys showed a similar condition to those of the brain. The spleen appeared normal, except for the large number of white blood cells. These appearances will be described in detail in a later part of this bulletin.

The following places were visited and a few cases will be described from each outbreak:

HOWARD, LA.

(August 27, 1906.)

Case 1—Mare, in good condition. This case had the following history: She was first noticed weak and stumbling blindly about. The owner said that she was breathing badly and had high fever, though he did not use a thermometer. When seen by the writer she was lying broadside in a comatose condition; respirations regular and deep, as in sleep; pulse slightly weak and accelerated; temperature 102.5° F.

Case 2—Mare about to foal, was in the first stage of the disease; was slightly blind and was so weak that she would stagger if moved. Temperature 104.5° F. Respiration and pulse much accelerated. She was still able to eat and drink. This was 8 o'clock in the evening. The following morning she had foaled a living colt, was unable to rise, but continually struggled to do so. She was unable to stand when assisted to her feet; quite blind and delirious. Temperature 100° F. Respirations and pulse slightly increased. Visible mucosæ anæmic.

Case 3—This was a post-mortem upon an animal belonging to the same owner, as Case 2. She died the night previous.

Brain—Blood vessels of the brain and meninges injected. Much effusion in the sub-dural space. Cross sections of the brain and medulla showed numerous areas of petechiæ and blood extravasations. No softening or change in consistency of the brain substance could be made out. The trachea contained much exudate of a very offensive nature. Lungs were hepatized, especially the lower one. The blood was dark and frothy and did not clot readily. The heart was normal in appearance but contained anti-mortem clots in both halves extending from the auricles through the ventricles into the aorta and pulmonary arteries for three or four inches. The spleen and liver contained much blood. The kidneys showed slight congestion.

Case 4—This was the last of the above owner's animals. He had already lost two, one was very sick and as we were about to leave he led up his last one, already partially blind, very weak and showing every symptom of the disease. Pulse 60, respirations 25, and temperature 105.5° F.

LOCKPORT, LA.

The disease had been raging in this vicinity on both sides of the bayou (Lafourche) for several weeks. The following cases will be mentioned:

Case 1—A large gray mare, in good condition, was attacked Tuesday morning; in the evening was given a purge, with no appreciable effect. On Wednesday she fell and was unable to rise afterwards. Thursday night we saw her, a few minutes before she died. At this time the pulse was very rapid; respirations labored and temperature sub-normal. This animal was used in a livery establishment and was first noticed ill as above stated. The first symptoms were drowsiness, weakness and persistent turning in a circle, always toward the same side. She rapidly grew weaker and finally fell. Her feed had been timothy hay and oats. No grass could be accounted for, unless allowed to eat while on a trip.

Case 2—A doctor's driving mare became affected about the same time as the above animal, but did not have so severe an attack. Instead of being comatose she was delirious. At the time we saw her she was convalescing, but very weak. Respirations and pulse normal. Temperature 97° F. Her treatment had consisted mainly of stimulants. Later it was reported that she had recovered. Feed was hay and oats.

Case 3—Gray mare, in fair condition, was taken sick at 10 o'clock Thursday night. The following morning her temperature was 104.8° F. Pulse and respirations slightly accelerated. Very weak and stupid. Ice had been applied to the poll and back during the morning. She grew rapidly worse and was unable to stand after a few hours. An aloes purge was administered, with no apparent effect. Died Saturday morning. Feed had been corn and grass. The corn was native grown, and was from the same crib from which she had been fed all the previous winter and spring.

Case 4—Bay horse, convalescing. This animal had suffered a mild attack, was never unable to stand and at this time was able to take some food and water. Feed hay, corn and grass.

Case 5—A large mare, in fine condition. This case was across the bayou from the preceding animal. She was taken sick the previous night; was very uneasy and persisted in turning in

one direction. She became rapidly worse and fell about day-break, unable to rise again. At 10 o'clock in the morning she was lying broadside, in a semi-comatose condition; pulse and respiration normal. Temperature 103° F. Feed consisted of hay and corn, such as she had been fed all the year; no grass, except what she picked to and from work. This owner had lost one or two horses with a similar disease a few months previous.

Case 6—Horse, taken sick Saturday morning. In the afternoon his pulse and respirations were practically normal. Temperature 104° F.; was very weak and would fall whenever moved. Feed was corn and forage.

Case 7—Horse, became affected at 10 o'clock in the morning. Led home a distance of ten miles. At 5 o'clock in the afternoon temperature 104.8° F. Pulse 45. Respirations slightly labored and accelerated. He was very weak and drowsy. A purge was administered, also two drachm doses each of potassium bromide, quinine and belladonna every three hours. It was reported that he survived. Feed: hay, corn and a small amount of forage.

Case 8—A two-year-old filly, taken Friday evening. She was first noticed very weak and drowsy. At 10:30 o'clock the following morning her temperature was 101.4° F. Breathing and pulse normal. She was very weak and presented a very typical case of the disease. Feed was entirely of grass.

THIBODAUX, LA.

Several animals were studied in this vicinity. The following case will be given in detail. Several post-mortems were made. The results of two typical ones will be included.

Case 1—This was a young high-strung saddle horse. He was used one-half day, Monday, and nothing wrong could be detected with him. The following day at noon he was noticed sleepy and drowsy, though he had been playing in the stall during the forenoon. He was then given one pint of raw linseed oil, nothing else being done except to keep him cool, clean and quiet until Thursday, when a local veterinarian was called. At this time the temperature was 105° F. Ice was used upon his spine and head. July 6, 3 p. m., he fell, after being sick sixty-three hours, but after resting an hour or so he recovered so far as to be able to rise, but soon fell again. At this time the

temperature was 104° F.; pulse 58; respirations 36. His temperature was never less than 104, often as high as 106, and just before he died (Saturday night at 8 o'clock) his temperature reached 108° F. Respirations 72. It was impossible to count the pulse. The owner said that he had never seen a horse suffer so much during the last hour. He was sick four days and fourteen hours. During this time he never lost consciousness. Liniments were tried over the brain, but they produced so much excitement that they had to be removed. His feed had been oats, bran, alfalfa and green corn. No grass in the previous four weeks.

Post-Mortem No. 1—Mare, in good condition; sick about three days.

Heart—Normal, in diastole. Blood dark and tarry; did not clot readily. Lungs slightly discolored, especially the lower one.

Brain—The blood vessels of the brain and meninges showed much congestion. Cross sections of the brain substances showed many petechiæ and blood extravasations. No softening or other conditions were detected.

Liver—The liver contained much blood, which was thin and showed an abundant black precipitate.

Spleen—Weight 1½ pounds. Was white and bloodless. It was with difficulty that blood could be obtained for films.

Kidneys—The kidneys were slightly congested. Weight 1½ and 2 pounds.

Digestive Tract—Normal. Contents were in a soft condition, except in the posterior part, where it was dry and hard. Stomach contained only a little colorless fluid.

Post-Mortem No. 2—Bay gelding. Sick two and a half days. Blood slightly darker than normal and did not clot readily. Heart normal. Pericardium slightly congested. Lungs normal, except a slight congestion on the inferior side.

Brain—There was much congestion of the blood vessels of the brain and meninges, but no change in consistency. The substance of the brain was ecchymotic.

Liver—Weight 24 pounds. Dark in color and slightly congested.

Spleen—Very much darkened. Contained much blood and weighed six pounds.

Kidneys—Some slight congestion. Weight 2 pounds and $2\frac{1}{2}$ pounds.

Digestive Tract—Stomach and other organs apparently normal, except the posterior part, which contained hard, dry feces.

BATON ROUGE, LA.

Case 1—Post-mortem of a four-year-old mare. This animal was first noticed sick in the evening. Died the following day at 1.30 p. m. The post-mortem was made one-half hour after death.

Brain—The blood vessels of the meninges and brain substance were much injected. The brain substance showed congestion and cross sections showed numerous petechiæ and ecchymosis. The medulla had the same appearance. No change in consistency could be detected. The cord in the lumbar region was normal. Trachea and lungs normal. Blood dark. Heart normal in size and appearance. Both ventricles contained anti-mortem clots.

Stomach—Normal in appearance. Contained much sour feed and grass.

Small Intestines—The intestines appeared normal from the exterior, but when opened showed pronounced inflammatory conditions of the posterior fourteen to sixteen feet. The mucosa was much inflamed and easily detached. The intestinal contents was a watery blood-colored liquid. The large intestine was normal, except for a large amount of dry feces.

Kidneys—Normal in size and slightly congested. Bladder empty.

Spleen—Normal in size and contained much blood.

PATHOLOGICAL HISTOLOGY.

Horse No. 1—All organs removed within one hour after death. Brain fixed in formalin; all other tissues in sublimate.

Brain. Except for a shrinkage of certain of the nerve cells there was but little change in the nervous tissue. The blood vessels of the pia and arachnoid and of the brain proper were filled with blood. In the larger vessels especially, numerous leucocytes, mostly of the polynuclear variety, were seen within the vessels, passing through their walls and in the tissues surrounding the vessels. (Fig. 2)

The smaller vessels showed the same general conditions, except the vessels were shrunken, and red and white cells had in many cases passed into the perilymph space. Few leucocytes had, however, invaded the tissues from the small vessels. In some areas many polynuclear leucocytes were present in considerable numbers throughout the section. Few red cells have been found without the perilymph space.

Sections of the hippocampus showed the blood vessels to be in the same condition as in the other areas of the brain.

Kidney. Considerable hyperæmia was present, also a slight degeneration and desquamation of the epithelial cells of the tubules.

Liver. The inter and intra-lobular veins contained much blood, in which were many leucocytes, mostly of the polynuclear variety. These leucocytes were also found in the tissues surrounding some of the larger blood vessels and even passing through their walls. (Fig. 3.)

Intestine. Portion found congested at a post-mortem examination. The coats of the intestine were thickened, especially sub-mucosa. There was a slight hemorrhage in the mucosa and congestion of the vessels of the mucosa and sub-mucosa. Considerable fibrin was seen in the sub-mucosa. Leucocytes were present in fairly large numbers in the mucosa. Many of these were of the polynuclear variety. (Fig 4.)

Spleen. Nothing abnormal except a considerable number of polynuclear leucocytes in the pulp.

Brain No. 2—Sublimate fixation. Animal sick four or five days. Brain removed 18 hours after death. The blood vessels showed the same conditions as those described in Brain No. 1. Post-mortem changes, however, were so great that other conditions could not be studied.

Kidney No. 2—Horse sick three days. Kidney removed six hours after death. Hyperæmia not so marked as in Case 1, although the larger blood vessels were congested. (Fig. 5.) The glomeruli were congested. A moderate amount of fibrinous exudate was found between the tubules and granular degeneration and desquamation of the cells of the tubules was quite marked. The capillaries were also congested. Figure 6 shows the degeneration of the tubules.

BACTERIOLOGICAL INVESTIGATION.

Cultures were made from the brain, liver, spleen and heart from a number of animals, but no growth took place except in one case, in which there was evident contamination, as only the pus organisms were recovered. Smear preparations were also made from the various organs, but no bacteria could be found, except in one case a gram-negative diplococcus was found in the exudate from the brain. The smears were stained for both bacteria and protozoa.

BLOOD EXAMINATION.

Complete examinations were made in only three cases. The blood was collected from an ear vein or a piece of the ear clipped off with scissors. In either case care was used to have the place free from hair, dry and clean. Gower's hemoglobinometer was used for the hemoglobin content. The blood was used in a dilution of one to one hundred in Toisson's fluid. Both red and white cells were counted in the same preparation in a Zappert-Ewing counting chamber. One hundred squares were counted for the red, the whole rules space for the white. Two counts were made and an average taken, unless great variation occurred, when other counts were made.

BLOOD COUNT.

No.	Hb.	Reds.	Whites.	Leucocytes.	Poly's.	Eosin's.	Mono-nuclears.	Mast.
1	60	5,304,000	8,500	33.8	52.1	1.3	12.1	.7
2	90	5,676,000	10,600	13.1	73.9	12.5	.5
3	62	6,840,000	12,330	30.1	59.9	2.	6.6	1.3

REMARKS.

No. 1 had been sick several days with a mild type of the disease. It later recovered.

No. 2 had been sick but 36 hours and died a few hours later.

No. 3 had been sick 10 hours and died twelve hours later.

INOCULATION EXPERIMENTS.

In all nine animals were inoculated—four rabbits, four guinea pigs and one horse. The animals were all adults and the inoculations made sub-cutaneously. The blood used was drawn aseptically from the jugular vein into sterile test-tubes and defibrinated under as aseptic conditions as possible. The citrated

blood was about three parts blood and one part citrate solution (sodium chloride 0.85 per cent and sodium citrate 1 per cent). The brain emulsion was made by grinding a portion of the brain substance in sterile distilled water. The source of material was, for inoculations 1 and 2, from a mule a few hours before death; for 3 and 4, from a horse with a mild attack of the disease and which later recovered; 5 was from a horse sick for three or four days and died two days later; 6 and 7, from the brain of the same animal as above, 10 hours after death; 8 and 9, from the brain of a mare, removed one-half hour after death and injected two hours later. Inoculations 6 and 7 were made at the place of the post-mortem.

TABLE OF INOCULATIONS.

No.	Date.	Animal.	Material.	Source of Mat.	Dose.	Remarks.
1	Sept. 10	Rabbit	Defibrinated Blood	Mule	1 cc.	Survived
2	Sept. 10	G. Pig	Defibrinated Blood	Mule	1 cc.	Survived
3	Sept. 20	Rabbit	Citrated Blood	Horse	1 cc.	Survived
4	Sept. 20	G. Pig	Citrated Blood	Horse	1 cc.	Survived
5	Sept. 24	Horse	Cit. Blood and Defib. Blood	Horse	1 cc. each	Survived
6	Sept. 26	Rabbit	Brain Emulsion	Horse	2 cc.	Died Oct. 10
7	Sept. 26	G. Pig	Brain Emulsion	Horse	2 cc.	Survived
8	Sept. 26	Rabbit	Brain Emulsion	Horse	2 cc.	Survived
9	Sept. 26	Pig	Brain Emulsion	Horse	2 cc.	Survived

No. 6 died October 10, very suddenly. Post-mortem showed marked emaciation. Brain and other viscera perfectly normal. Several animals in the pen from which this one was taken died in about the same manner and gave the same post-mortem appearances. Therefore we believe that the inoculation had nothing to do with this death. None of the animals showed any effect of the inoculation either by sickness or abscess formation at the point of inoculation.

CONCLUSIONS.

1. The cause of the disease or its infective nature has not been determined by these investigations. No micro-organism has been encountered that would account for the disease.

If we accept the statements of the different owners, regarding the character of the materials consumed by the different animals as absolutely accurate then the theory of food contamination does not seem to be well taken for the following reasons:

(a) The low per cent of animals affected under exactly similar conditions.

(b) The widely separated cases.

(c) The fact that the animals attacked were not confined to any particular feed or combination of feeds.

2. The pathological findings pointed out changes in the blood vessels of the brain and meninges. Degeneration and some other changes were present in some of the internal organs. Just how much of these pathological changes are due to the disease, and how much to the post mortem changes, is hard to say. In this climate post mortem changes take place so rapidly that it is difficult to get suitable material for study. However, in case (1) one the material was all removed and fixed within one hour after the death of the animal.

NOTE.

During the outbreaks recorded by this bulletin, our time was occupied chiefly in the field, endeavoring to discover and suggest immediate remedial and preventive measures; in consequence, more thorough and systematic investigations were interfered with, which, however, we intend to continue when the opportunity presents itself.

AS TO PREVENTION.

Although the exact cause of meningitis in horses and mules has never, as yet, been satisfactorily demonstrated, either in this country or abroad, it has been the opinion of Dr. W. H. Dalrymple, of this Station, who has experienced several previous outbreaks in Louisiana, during both the spring and summer months, that the cause was, in some way, associated with the condition of the feeding materials—either grasses or cured products, such as corn, etc.—brought about by the attack of molds or fungi; and that when a complete change, to food that was absolutely sound, was made, the disease was either checked, or disappeared entirely.

This, also, would seem to have been the experience of other investigators.

Consequently, until the exact nature of the agent producing meningitis, as well as a possible remedy, has been discovered, we would urgently recommend to stock owners, that, as soon as they observe the first symptoms of so-called "staggers," they at once make a change from feeding materials that are at all suspicious, to those that are perfectly sound. Or, as a matter of prevention at all times, that they do not supply to their animals, or permit them to consume, food of any kind that is not absolutely sound and free from molds or fungi.

REFERENCES.

1. McCallum and Buckley—Acute Epizootic Leucoencephalitis in Horses. Bulletin No. 80, Md. Agr. Exp. Station, 1902.
2. McCarthy and Revenel—A Pathology for Forage Poisoning, or the So-called Epizootic Cerebro-Spinal Meningitis of Horses. Journal of Medical Research, Vol. 1 (1903), p. 243.
3. Mayo—Enzootic Cerebrites or Staggers of Horses. Bulletin No. 24, Kansas Agr. Exp. Station, 1891.
4. Moore—The Pathology of Infectious Diseases of Animals.
5. Law—Veterinary Medicine. Vol. IV.

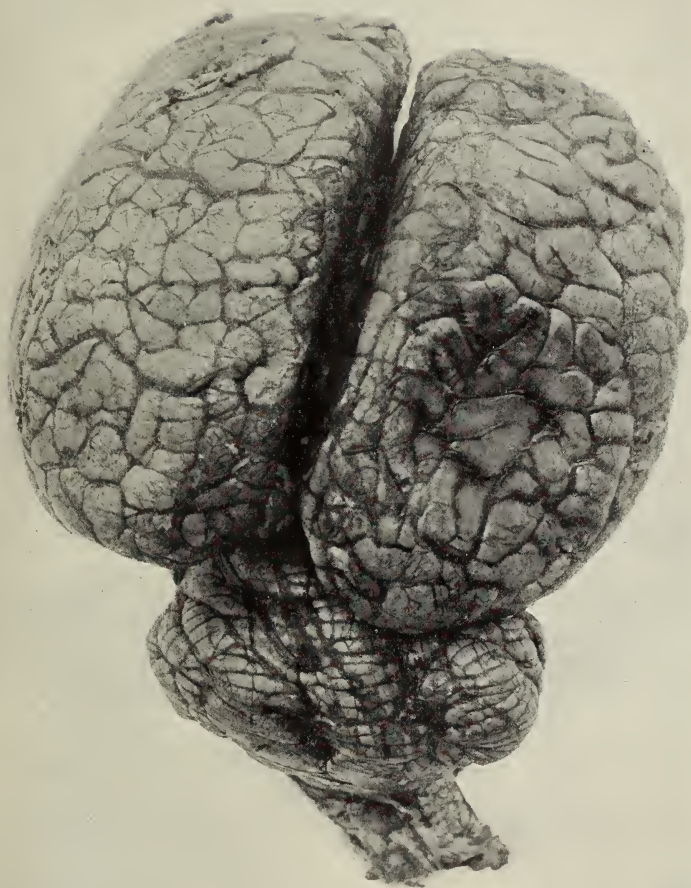


FIG. 1.—Brain of horse. Note the injection of the blood vessels.



FIG. 2.— Photograph of section of a large vessel of the brain. Note the blood within the vessel and the numerous leucocytes passing through its walls and in the tissue adjacent.

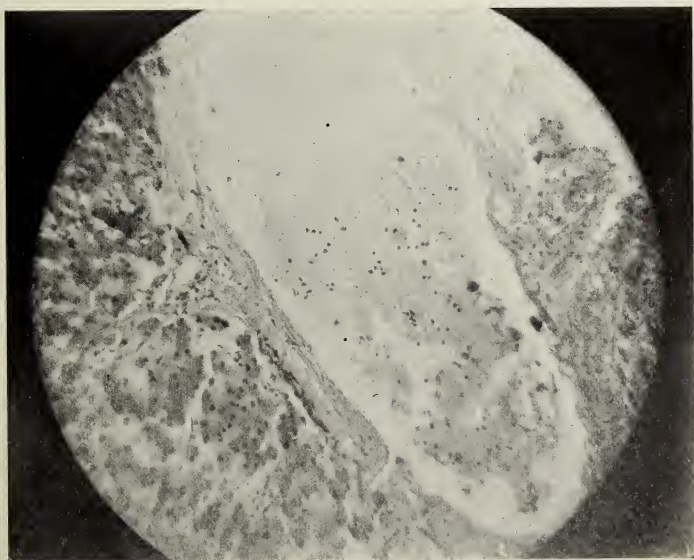


FIG. 3.— Photograph of section of liver of a horse through a large blood vessel. Note the large number of leucocytes within the vessel.

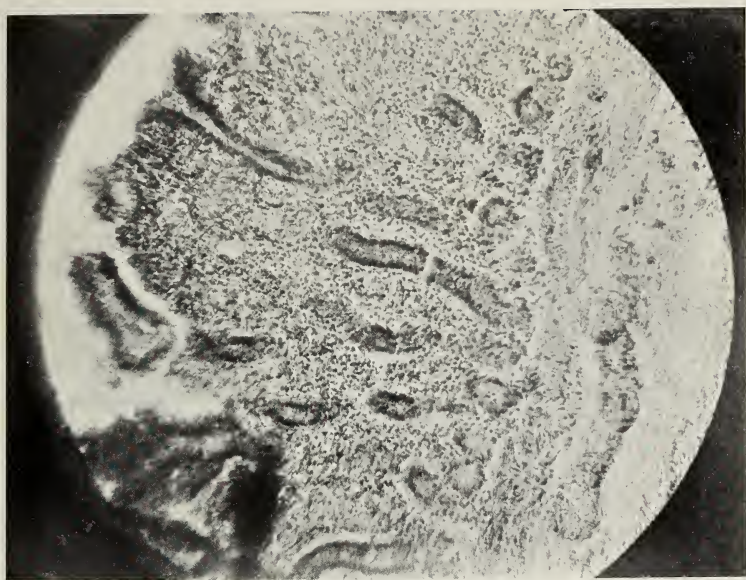


FIG. 4.—Photograph of section of intestine of horse showing large number of leucocytes in mucosa.

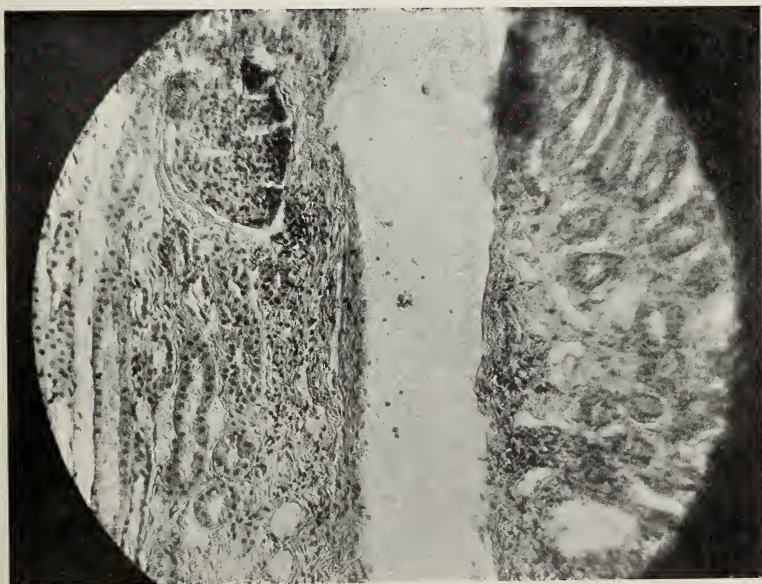


FIG. 5.—Kidney of horse showing congestion of the large blood vessels.

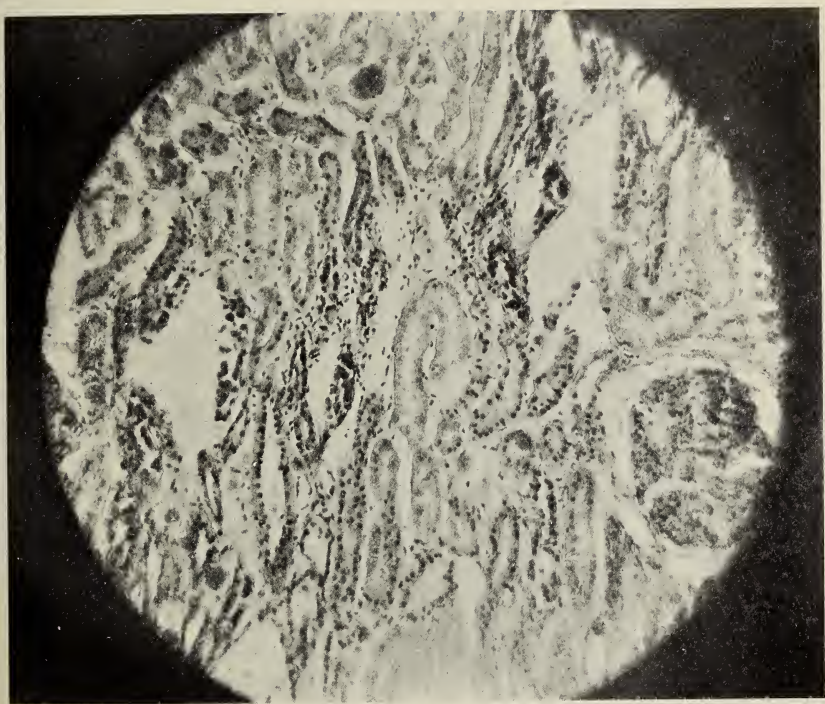


FIG. 6.—Kidney of horse showing degeneration of the tubules. The tubules to the right and in the centre show the condition especially good.

